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THE CONDITION OF THE HEART IN ANÆMIA, AND THE CAUSE OF THE PULMONARY MURMUR.

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THE subject which I wish to bring forward is the cause of the pulmonary systolic murmur, so constantly present in anæmia. And in order to give the grounds for the views I hold upon this question, it is necessary for me to go more fully than I otherwise should have done into the general question of the condition of the heart in anæmia. Some of the following observations are capable of direct proof; and in support of the rest much evidence may be called, though it falls far short of proof.

1. Anæmia is a frequent and sufficient cause of fatty degeneration of the heart and of dilatation of its cavities.

Simple anæmia is but rarely fatal. I can, therefore, offer no post-mortem evidence from my own observation as to the condition of the heart-muscle under these circumstances. However, Walshe, Ormerod, Virchow, Ponfick, and many others all testify to the influence of anæmia in producing fatty degeneration of the heart.

Dr. Goodhart (*Lancet*, vol. i., 1880) expresses the view that in all forms of anæmia in women the heart's impulse becomes diffused and displaced outward; the heart-muscle undergoes fatty degeneration; and the heart, especially the left ventricle, becomes dilated. And he has demonstrated post-mortem in five instances that simple anæmia from loss of blood, in adults of both sexes and of very varying ages, is constantly associated with fatty degeneration of the ventricular walls and dilatation of the cavities, especially the left.

Dr. Bristowe says: "Dilatation, which is sometimes the primary change in hearts that are simply feeble, not only impairs efficiency, but actually furnishes an incentive to cardiac exertion and overgrowth."

2. In a definite class of cases of anæmia, including chlorosis, cardiac dilatation is a constant and important condition which urgently requires more general recognition and treatment.

Very little space is devoted to this subject in most of the text-books, and but little importance is as yet attached to it. Consequently, like many others, I was left to find it out for myself; but when once my attention had been directed to it, I found that it had been dealt with in several special articles.

During the last four years I have carefully examined into the state of the heart in five-and-thirty cases of simple anæmia, chiefly in young females, that have been under my care in the Nottingham General Hospital. Each case was under observation for several weeks, and a few for a much longer time. And during the same period I have made similar observations in a much larger number of cases of anæmia due to various cachectic conditions. And as a result I am able most confidently to confirm Dr. Goodhart's statement, at any rate so far as to say that in chlorosis and the simple anæmia of young women the heart's impulse becomes diffused and displaced outward almost invariably. But, contrary to his experience, I think the clinical signs point in these cases to the dilatation affecting chiefly the right side.

3. In such cases the right side is often the one chiefly affected.

I have come to this conclusion because the heart's impulse is invariably displaced outward, but generally little, if at all, downward. The apex-beat (that is, the part of the impulse farthest to the left and downward) is, in the vast majority of cases, in the fourth space, sometimes in the fifth, but I have never found it in the sixth. In horizontal positions the apex is usually in the nipple-line, or a little outside it, but it varies from a half inch inside to one and a half inches or more beyond it. To compensate for the variable position of the nipple I have always made in addition actual measurements, and have found the apex-beat from three and a half to four and three-quarters inches, or further, from mid-sternum. The most forcible pulsation is often in the third space, about one inch inside the nipple-line; and I attribute it to the impact of the middle or upper part of the anterior wall of the right ventricle, and not to the auricular appendage. Rindfleisch,[1] in speaking of hypertrophy of the right ventricle, says: "The heart's apex is no longer formed by the left ventricle alone; it is partly, or even wholly, formed by the right one. The apex-beat often becomes indistinct; in its stead we find a basic impulse due to the contact of the conus arteriosus with the chest-wall during each systole, in consequence of the enlargement of the base of the heart in its antero-posterior diameter."

In well-marked cases, especially those occurring acutely and to which over-exertion has contributed, there is often a systolic murmur with its point of maximum intensity over the fourth left costal cartilage. This, I take it, indicates regurgitation through the right auriculo-ventricular orifice. And this view is confirmed by the turgid condition of the external jugulars, especially the right, which pulsate synchronously with the heart and fill from below.

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[1] Pathology. Sydenham Society's Translation. Vol. i. pp. 270, 271.

4. There are many forms of anæmia, in some of which there are heart-murmurs, and in some not; in some the heart is enlarged and dilated, and in others not. Where the heart is dilated, several different conditions contribute to bring about this result. There are several different murmurs in anæmia, one or all, or none, of which may be audible in a given case. Hence, partly, the divergence of opinions.

Typical chlorosis I believe to be in the first instance largely, if not entirely, a developmental disease, and, hence, occurring at or about puberty, before development is completed. In one class of cases, severe in point of intensity but limited in numbers, the essential lesion is a "hypoplasia or congenital narrowness of the aortic system." This view, originally expressed by Virchow, has been confirmed by Rokitansky, Bamberger, Quincke, and recently by Fraentzel. In the *Practitioner* for 1889 I recorded a well-marked example, together with the post-mortem conditions. The heart varies much. At times when the general development is poor and there has been no undue bodily exertion, and the affection has lasted but a short time, the heart is abnormally small, corresponding to the dimensions of the aorta, like the heart of a child. At other times, especially when there has been rapid body-growth, underfeeding, and a call for over-exertion, the heart is enlarged, dilated, and hypertrophied. "The vascular walls and their contents are both derived from the same mesoblastic tissues of the embryo. Let us suppose the unborn defect of developmental energy to extend not merely to the blood, but to the cellular lining of the vascular tree as well. A hypoplasia of the vascular system will then be inevitably associated with the hypoplasia of the blood."

In many, indeed in the great majority of cases of chlorosis, the full development of the aortic system is only *delayed;* and the patients eventually get quite well. The ratio of the different organs to one another varies in different individuals, and only approximately approaches a perfect type. In some the brain and nervous system predominate; in others the chest and respiratory system are unusually well developed. Some have a large bony framework; and yet in others, the liver or the kidneys are always "inadequate."

Between the ages of twelve and fifteen in the female, growth and development proceed with extraordinary rapidity; stature and weight being both actually and relatively greater than at corresponding ages in the male. In some cases the development of the vascular system may lag behind the requirements of the body. With this condition, or even with only the feebleness of rapidly growing tissues, much physical exertion (in maid-servants in running up and down stairs; in girls of a better class in playing tennis, etc.) may result in cardiac dilatation.

It is well known that the development of the various organs does not proceed *pari passu*, and that various causes may disturb or delay its progress. I have seen the uterus of a girl of seventeen, who had died of phthisis, indistinguishable in point of size and shape from the uterus of a nine months' fœtus placed beside it, and only to be recognized by the condition of the ovaries. In some cases gastric ulcer or persistent gastric catarrh, in others want of fresh air, may by interference with general nutrition, lead to a similar delayed development of the arterial system, and through that to cardiac dilatation. It is well known that in some forms of heart affection occurring in early life, more especially in mitral stenosis, the aorta, and the arterial system generally, remain imperfectly developed. There is a general hypoplasia of the aortic system. I have myself met with many well-marked instances. Balfour says:

> "There is every reason to believe that mitral stenosis is by no means infrequently congenital . . . . and that in these instances it is invariably associated with hypoplasia of the aortic arterial system, and consequently of the body generally."

And again:

> "But should a rheumatic attack in childhood or early youth be followed by any considerable mitral stenosis, the effect of this must be to accumulate blood behind the stenotic valve, to diminish the wave of blood sent forward, and thus to lessen the normal distention of the aorta, and to impair the nutrition of all the tissues, producing that retarded development so well known as the constant result of serious cardiac disease occurring in early life, technically termed hypoplasia, affecting primarily the aortic arterial system, and secondarily all the organs of the body."

Dr. Clifford Allbutt has called attention to the influence of underfeeding in producing degeneration of the heart and consequent dilatation. This, of course, applies to both sexes and all ages, but it is specially applicable to chlorosis, in which condition the capriciousness and perversion of the appetite are notorious. But in a far larger number of cases, in which an accusation of capriciousness or perversion of appetite could hardly be sustained, it is a noticeable and very important fact that these anæmic girls take a dislike to the more highly nitrogenous forms of food—meat, fish, milk—and habitually omit them from their dietary. It is probable that the undue prominence of the carbohydrates, and the comparative absence of the albuminates in the food, has at least as much influence in producing the *embonpoint* so general in chlorosis, as the want of exercise and deficient oxygenation of the blood and oxidation of the tissues to which it is so generally attributed.

It is now widely recognized that in many forms of anæmia, though by no means in all, and possibly not to a well-marked degree in all the examples of the same form, there is a distinct rise in the vascular tension. This is readily perceived by the finger on the radial pulse; but it is still more distinctly shown by the use of the sphygmograph. I have several tracings which show a distinct rise in vascular tension, and many others which show a tension quite up to the normal. These tracings are of the more value because one of the commonest complications in the interpretation of sphygmographic tracings is practically eliminated in these young patients, viz., rigidity and degeneration of the walls of the vessels. The increased tension is so contrary to one's preconceived ideas that it was very slow in gaining acceptance. The causes of it, and more especially its presence in some cases and its absence in others, are not easy of explanation.

It has been shown by Eberth and Schimmelbusch, and more recently and more completely by Professor Hamilton, of Aberdeen, that alterations in the blood may result in a considerable impediment to its free circulation. In healthy blood, the specific gravity of the colored corpuscles is nearly alike with that of the plasma, while that of the colorless is considerably less. Consequently the colored corpuscles keep mainly in the axial stream, while the colorless keep largely in the periphery. Any alteration in the constitution of the blood, either in the plasm or in the colored corpuscles, which form the enormous majority, such as to make the corpuscles either specifically lighter or heavier than the plasma, causes them to move less in the axial and more in the peripheral stream, makes them touch and roll along the sides of the vessel, and very greatly increases the amount of friction, which consequently entails a corresponding increase in the propelling power. There is much reason to suppose that in some forms of anæmia such alteration in the relative specific gravity of the corpuscles and the plasma does occur. In the majority of these chlorotic forms of anæmia the red corpuscles are little, if at all, reduced in number, but are very deficient in hæmoglobin.[1] That makes it extremely probable that they are also reduced in specific gravity, but I believe that point has not been proved yet. Hence, in all probability, the high-tension pulse of anæmia, and, to some extent and in certain cases, the consequent dilatation and hypertrophy of the heart.

The anæmia of malignant disease; of phthisis, of cachectic conditions, and of chronic wasting diseases, is more complex. The total volume of the blood is diminished. The corpuscles and the plasma suffer more nearly in equal degree. There is less capacity for exertion, and so less strain is thrown on the heart. The general nutrition is more gravely impaired. The heart has insufficient pabulum, and so atrophies in correspondence with the general wasting of the body. In these cases, however profound the anæmia, there are generally no cardiac murmurs.

In post-febrile anæmia I have already elsewhere[2] referred to the frequency of cardiac degeneration and dilatation, and to the variability of the murmurs.

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[1] AMERICAN JOURNAL OF THE MEDICAL SCIENCES, May, 1890.

[2] Provincial Medical Journal, July, 1890, p. 395.

In cases of anæmia of recent origin, where there has not been sufficient time for dilatation and enlargement of the heart to ensue, there are generally no cardiac murmurs. I have noticed this on many occasions. I may quote two examples:

An unmarried girl, aged eighteen, who had been confined in the workhouse, was shortly afterward admitted into the general hospital with the most profound anæmia, but there was no trace of a cardiac murmur, and no evidence of dilatation or enlargement. She soon got quite well.

A married woman, aged twenty-two, was admitted suffering from most marked anæmia of recent origin, due to metrorrhagia and over-lactation. There was no trace of a cardiac murmur or of enlargement of the heart, and the pulse tension was low. She soon recovered completely.

In these and similar cases the absence of enlargement of the heart may be in part due to rest and absence of exertion from the commencement of the anæmia; and to general failure of nutrition, tending to atrophy of the heart; as well as to the recent origin of the anæmia.

In what I may call, for want of a better term, "gastric cases of anæmia," dilatation of the heart and cardiac murmurs are slight or absent. By "gastric cases of anæmia," I mean those which commence with or are early associated with gastric ulcer or chronic gastric catarrh. In these the general nutrition usually fails, and the state of the heart is one of atrophy rather than of hypertrophy, and hence cardiac murmurs are absent. In these various forms of anæmia where the heart is dilated or hypertrophied, or both, I conceive the change to be due to one or more of the following causes:

1. Congenital narrowing, or delayed development of the aorta, and the aortic arterial system, requiring greater pumping power to supply the needs of the circulation.
2. Increase of resistance in the peripheral part of the circulation from unequal changes in the specific gravity of the component parts of the blood. (These two classes include most of the cases of high-tension pulse.)
3. Malnutrition and degeneration of the cardiac muscle from the circulation through its walls of blood deficient in some of its proper constituents. A larger bulk of this flabby, degenerated, or relaxed muscle[1] is required to do the same amount of work as a smaller bulk of normal muscle in good tone will do. Hence hypertrophy rapidly follows on dilatation.

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[1] Balfour: Diseases of the Heart, p. 156. "In chlorosis, and after fevers, etc., the change in the condition of the heart consists in such a complete relaxation of its tissues that in its most extreme form it seems as limp as a piece of wet paper."

4. Overwork, especially associated with constriction of the chest and waist, as is usual in females. (Constriction of the chest during exertion apparently favors cardiac and arterial disease in soldiers.)

There are many different cardiac murmurs commonly audible in anæmia, but, so far as I know, they are all systolic in rhythm. The commonest, and the one most typical of anæmia, is that with its point of maximum intensity in the pulmonary area. The cause of it I shall discuss later.

The next most frequent murmur is at the left apex, and is a true regurgitant murmur, due probably in most cases to relaxation and dilatation of the left ventricle and relative incompetence of the mitral valve. Rheumatism is very common in anæmia, and in some of these cases there is probably rheumatic valvulitis.

A systolic murmur is not uncommon, too, in the tricuspid area, due in all probability to dilatation of the right ventricle and relative incompetence of the tricuspid valve.

A systolic murmur over the aortic cartilage is, in my experience, rare in these cases, and is then often permanent and apparently due to organic disease, except in those instances where it is only communicated from the pulmonary area, and is not independent. These murmurs disappear[1] in the following order, viz.: tricuspid, mitral, aortic, pulmonary.

Thus, there are several distinct murmurs in anæmia, and this has led to much confusion in the discussion of their etiology. The most characteristic murmur is the pulmonary, and of that I shall have more to say shortly.

5. *The pulmonary, so-called hæmic, systolic murmur is not due directly to a change in the blood.*

There are three distinct classes of murmurs called hæmic, namely: the venous, the arterial (those produced at the orifices of the pulmonary and aortic arteries), and the purely cardiac (those audible over the mitral and tricuspid openings); and they have all been for many years attributed to an altered condition of the blood.

That this is a satisfactory explanation in the case of the venous murmurs most admit, although they may not agree as to the exact nature of the alteration. It seems probable that the change is more in the direction of diminished quantity than in altered consistence, and that the murmur is due to imperfectly filled vessels.

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[1] Balfour says: "All the ordinary murmurs are not infrequently found associated in chlorosis with a true murmur of mitral regurgitation distinctly and clearly audible in the mitral area, and accompanied by distinct accentuation of the pulmonary second sound. And these murmurs disappear in the exact order in which we should expect them to do so; first the mitral murmur vanishes, then the murmur in the aortic area ceases, next the auricular murmur disappears, and, last of all, the venous murmurs become inaudible."

The cause of the arterial murmurs is a more complex problem. I am not prepared to say that diminished volume of the blood plays no part, but it is not sufficient to explain all cases.

It has been stated by Richardson, as a result of experiments, that if an animal be bled until it is feeble, an anæmic murmur becomes apparent; *while if large quantities of saline solution be injected into the vessels*, so as to compensate for the loss, the murmur does not disappear. Much has been founded on these and similar experiments to prove that the altered condition of the blood has, in some way, to do with the generation of the abnormal sound in man. But it is never safe to reason directly from animals to man. And, fortunately, we have in diabetic coma the opportunity for a direct experiment on man to elucidate the point in question. Recently, in a case of diabetic coma in a woman aged twenty-five, Dr. Dickenson introduced into the veins of the arms 106 ounces of saline fluid in the course of one and a half hours. Next day, the patient having relapsed into a state of coma, 350 ounces were allowed to flow from a funnel into the veins of the leg. The patient recovered consciousness completely after three-quarters of an hour, but ultimately died. Within the space of thirty-two hours 456 ounces of saline fluid had been introduced into the veins. The *heart-sounds were feeble, but there was no murmur either in it or the great vessels.* And yet the blood must have been very hydræmic.

Further, in a case of pernicious anæmia lately under my care, in a boy aged sixteen, with lemon coloration of the skin and conjunctiva and abundant retinal hæmorrhages, and where the colored corpuscles fell at one time as low as 154,655 per c. m., the pulmonary systolic murmur was never loud and distinct, and was, during a great part of the time, difficult to catch.

6. The characteristic of the so-called hœmic, pulmonary murmur is that, though audible frequently in all the areas, its point of maximum intensity is in the second or third left interspace (usually the second), close to the sternum; and that it invariably becomes much less loud, and freguently totally disappears in the erect position.

In the early part of 1888, I was much surprised at the sudden disappearance of one of these pulmonary murmurs.

The patient was an anæmic maid-servant who had been admitted into the General Hospital, Nottingham, on account of syncopal attacks. There was well-marked dilatation of the heart, and a loud systolic murmur with its point of maximum intensity in the pulmonary area. The patient gradually improved, though the murmur did not alter much. She was allowed to get up. A few days later I examined the heart while the patient was standing. There was no murmur to be heard, and I congratulated myself and the patient on the rapid reëstablishment of a healthy cardiac state. A day or two later I happened to examine the chest again before the patient got up. The murmur had returned, and was as loud as ever. The thought suddenly struck me that the variability of the murmur was due to the influence of position. I made the patient stand, and the murmur became inaudible. On lying down again, it returned at once.

Since that date I have made hundreds of observations to test this point, and have full records of between thirty and forty cases of anæmia in which this phenomenon has occurred.[1] And the result of my experience is that in all cases of anæmia in which the pulmonary systolic murmur is present, it becomes much less loud in the erect position, and that in the majority it either totally disappears or leaves only an impure or murmurish first sound which might easily escape observation; but that the murmur returns at once on the patient assuming the recumbent position.

It is sometimes accompanied by pulsation in the episternal notch, and almost always by pulsation in the second, third, and fourth intercostal spaces near the sternum. The murmur is intensified by raising the patient's hips or the foot of the bed. It is unchanged by turning the patient on the left side. The murmur generally becomes much fainter when the patient lies on the right side, and *disappears when she turns on the face.* On the patient standing the heart is apt to palpitate, or at least become unduly accelerated. Under these circumstances observations are difficult. The murmur usually disappears, or, should it remain, it becomes difficult to differentiate.[2] After a few minutes the heart usually quiets down, and then more accurate observations can be made. It has been noticed by several observers that these pulmonary murmurs usually disappear during full inspiration,[3] especially if the patient holds the breath. An explanation of this will be suggested later. It is the contrary of what usually happens in apex-murmurs.

7. *This pulmonary systolic murmur, disappearing in the erect position, is due to the pressure of an enlarged, flabby, and dilated heart on the pulmonary artery.*

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[1] Gee says: "Murmurs audible in the horizontal position sometimes become much less loud and long in the erect—nay, occasionally altogether cease. This is most frequently the case with presystolic murmurs produced at the mitral orifice. The reason of the occurrence is not quite clear. I have not been able to find any statement in the text-books that this applies especially to the pulmonary, so-called hæmic, murmurs. Nor can I ascertain that such a view is at present the teaching of the schools. In fact, singularly little practical importance seems to be attached to the 'influence of position on cardiac murmurs,' although several monographs have been written on the subject."

[2] Gee states: "Murmurs are loudest sometimes when the heart beats forcibly, sometimes when it beats quietly."

[3] Gee says: "The loudness of murmurs is sometimes under the influence of breathing; thus, a systolic apex-murmur may be much louder during inspiration than during expiration.

On thinking over this strange variability of the anæmic pulmonary murmur, according to the position of the patient, a variability not found with anything like the same frequency or to the same degree in other murmurs, even the presystolic, it seemed to me that the only possible explanation of the murmur disappearing in the erect and in the prone positions, was that it was caused by the pressure of the heart—especially of an enlarged and flabby heart—on the pulmonary artery. This pressure may be increased somewhat during the systole by the "base-beat." I suppose the pressure to be communicated chiefly through the fibrous ring surrounding the orifice of the pulmonary artery. The counter-pressure would be partly the general fixity of the pulmonary artery by the passage of its branches into the roots of the lungs, and partly the arch of the aorta, in which the vascular tension is higher and therefore the vessel firmer, and which is held in position by the large branches coming off from it and by the general connective-tissue investment, This view somewhat resembles Russell's theory, with which I was unacquainted at the time, but the points of difference will be explained later on.

The explanations of the cardiac murmurs of anæmia have been many and various.

Marey supposed these bruits to be due to a diminution of arterial tension facilitating the production of sonorous veins at the arterial orifices. Clinical medicine convinces us that diminution of arterial tension is not sufficient to account for the production of a murmur; and, moreover, in many of these cases of anæmia we have seen that the arterial tension is raised.

Naunyn's theory, adopted by Balfour, is that the murmur is due to regurgitation through the mitral orifice into the left auricle, and that the point of maximum intensity of the murmur is not over the pulmonary artery, but one and a half inches or more to the left of the left edge of the sternum, where the appendix of the left auricle comes up from behind. In my own experience the point of maximum intensity has been, in the majority of instances, over the pulmonary artery, or over the conus arteriosus near the sternum, and not so far to the left as described by Naunyn and Balfour. I am not satisfied that there is any sufficient evidence that the left auricular appendage, unless in very exceptional instances, does come into contact with the chest-wall, but I believe as far as my own observations guide me, that in the vast majority of cases it is covered by lung. And, furthermore, the dilatation of the right ventricle so common in anæmia tends to push the left auricle still more to the back. But the strongest ground of all for rejecting this theory is, that in no small proportion of cases a systolic murmur, differing in character from the pulmonary one, can be heard at the apex, and is propagated for one or two inches toward the axilla, while the pulmonary murmur is audible in the usual position at the same time. The pulmonary murmur disappears on the patient standing erect, the apex murmur does not. And in many cases, after suitable treatment by rest, etc., the apex murmur disappears even in the horizontal position, while the pulmonary murmur remains for some weeks longer. This seems to me sufficient evidence of the independence of their causation. There can be little doubt that the apex murmur is due to regurgitation through the mitral orifice, in some cases from shrinking of the valve from valvulitis; in most, probably, from dilatation of the orifice, or from inefficient contraction of the muscles of the base (McAlister), or from general relaxation of the muscular tissue. In these cases, at any rate, the pulmonary murmur must be explained by some other cause, for it is unreasonable to suppose that regurgitation through the mitral orifice can produce two different murmurs at the same time, heard best in different areas, propagated in different directions, and one disappearing while the other remains.

Broadbent traces the cause of these murmurs to high arterial tension, owing to which the ventricle is unable to empty itself, and hence dilatation follows.

Bristowe (*Proceedings of the Medical Society of London*, vol. xi., 1888), speaking of the "murmurs often termed 'hæmic,' which are generally best heard about the sternal end of the cartilage of the left third rib," says:

> "How it is caused I do not pretend to decide. That it is directly due to altered quality of the blood I do not in the least believe. I should think it is connected with some want of due relation between the quantity of blood expelled from the ventricle in a given time, and the size or tension of the pulmonary artery, allowing of the development of coarse vibrations of the contents and walls of that part of the circulatory apparatus over which it is best heard."

Russel, while not disputing that the murmur heard in a large proportion of cases of debility, due to causes such as chlorosis, fevers, and parturition, is excited by mitral regurgitance owing to laxity of the ventricular walls, yet denies that its mechanism as expounded by Balfour is correct. It is not generally heard at the apex, the point where mitral murmurs are usually loudest, and it has not the purely soft blowing character of a murmur arising in this neighborhood. It also gradually disappears during inspiration. He explains its mechanism rather on the assumption that the left auricle becoming distended from the mitral regurgitation, presses injuriously on the pulmonary artery, and throws its blood into a state of sonorous vibration.

To this theory it may be objected that there is no evidence that the left auricle is distended in all the cases of anæmia in which the pulmonary murmur is distinct. As I have said before, in very many cases there is no apex murmur at any time, and in others it disappears before the pulmonary. In a large number of cases, therefore, the pulmonary murmur has to be explained in the absence of any evidence whatever of regurgitation through the mitral valve, and consequent distention of the left auricle. And in the very large number of cases of chronic heart disease from incurable mitral regurgitation this murmur is not present, notwithstanding that one may be assured that the left auricle must be dilated.

Furthermore, in mitral stenosis, when the left auricle is greatly distended, a systolic murmur in the pulmonary area, or in the third space, is exceptional.

Balfour says:

> "Not unfrequently, a systolic murmur with its point of maximum intensity in this situation (*i. e.*, an inch to the left of the left edge of the sternum in the second space) is due to the regurgitation almost always present in mitral constriction, many circumstances favoring in these cases the convection of this murmur upward to what we may term the auricular area, rather than its mere reduction outward into what is termed the mitral area. These being the great dilatation of the appendix auriculi, and the concomitant tendency of the left apex to become depressed within the chest by the distended right ventricle, which, in all these cases, is usually more or less *dilated*."

I believe that this murmur is not due to regurgitation through the mitral orifice, but to the dilatation of the right ventricle already alluded to, and to the weight and base-beat of the heart pressing on the pulmonary artery.

Balfour relates the case of a man with mitral stenosis, in whom there was a presystolic murmur audible at the apex, and a loud, rough systolic murmur in the pulmonary area. The systolic bruit became always very faint, and occasionally inaudible; on the patient assuming the erect posture, the presystolic bruit remaining unchanged. And he suggests that this was possibly because in this position the necessary compression of the pulmonary artery was prevented by the heart's weight dragging it down.

Quincke,[1] of Berlin, has related a case of mitral stenosis in which a pulmonary systolic murmur was present. He attributed it to hypertrophy without dilatation of the right ventricle, together with dilatation of the pulmonary artery, the murmur arising at the orifice of the pulmonary artery from the formation of a fluid vein. In this case the auricular appendix was thrombosed and lay deep in the chest; the auricular origin of the murmur from convection was therefore precluded. But the thrombosed auricular appendix would from its position readily become an instrument for compressing the pulmonary artery, especially when in the recumbent position a portion of the weight of the heart was communicated to it. I believe in this, as well as in the next series of cases related by Quincke, the essential condition is the dilatation of the right ventricle, and that the murmur is produced in some instances, perhaps, by the consequent *relative* stenosis of the pulmonary orifice; but in the majority by the actual stenosis of the pulmonary artery produced by the weight and upward pressure (base-beat) of an enlarged and flabby heart, communicated to the pulmonary artery through the fibrous ring surrounding the arterial orifice. All the conditions for this were present in Quincke's cases. Most of them were associated with debility. There was visible pulsation in the second left interspace, and there was an increase of the absolute cardiac dulness, which diminished *pari passu* with the disappearance of the pulmonary bruit. The murmur, too, disappeared when the pulmonary artery again became covered by lung, just as in chlorosis the murmur disappears during full inspiration. Quincke states that the systolic pulmonary murmur is accompanied by an uncovering of the base of the heart by retraction of the left lung from various causes, and he supposes that in these cases the systolic murmur is produced by compression of the pulmonary artery by the heart during its systole. The murmur is caused by the passage of the blood through the compressed and narrowed portion of the artery into the uncompressed and therefore comparatively dilated portion beyond, a state of matters which must be accompanied by the production of fluid veins at the part artificially narrowed; and which, therefore, if it exists, must be regarded as an efficient physical cause for the production of a systolic murmur. He bases his views as to the possibility of the production of this artificial narrowing, first, upon the fact that the pulmonary artery could in all these cases be felt and seen to pulsate. Quincke attributes much importance in these cases to the "uncovering of the base of the heart by retraction of the left lung from various causes." And Balfour emphasizes this still more in his cases. He says:

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[1] Balfour, p. 197. I have had no opportunity of referring to the original, Beiträge zur Enstehung der Herztöne und Herzgeraüsche, Berliner klin. Woch., 1870, No. 21, S. 249.

> "Now you will observe that all these cases agree in this, there was in every one of them retraction of the left lung, and uncovering of the heart and pulmonary artery, the exposure of the latter permitting its pulsations to be readily seen and felt. . . . The uncovering of the pulmonary artery also brings the valves nearer the surface; hence their closure is not only more easily felt, but also more easily heard."

This explains the *normal* sounds being more loudly heard, but does not account for the production of a murmur. I believe that in anæmia the uncovering of the heart and pulmonary artery, so far as it occurs, is due to the lung being pushed away by the enlarged heart. Where the lung is retracted from disease of its own tissues the heart gets more room, and is allowed more play, and hence without being enlarged or dilated may, in the recumbent position, fall back and press on the pulmonary artery sufficiently to cause a murmur. On this theory the disappearance of pulmonary murmurs, when the breath is held after a full inspiration, would be only partly due to imperfect conduction of the murmur from the heart and pulmonary artery being well covered by expanded lung, but largely also to the heart being better supported by the increased size and increased tension of the pulmonary air-cushion, and prevented from falling back.

Balfour speaks of the "auricular murmur as of such frequent occurrence in all febrile disorders, depending upon a slight degree of cardiac dilatation, and this has been occasionally, though rarely, accompanied by a murmur of similar origin audible in the mitral area." That febrile, or post-febrile cardiac dilatation should lead to a regurgitation through the mitral orifice causing a systolic murmur with its point of maximum intensity sometimes in the auricular area, and sometimes at the left apex, and sometimes equally audible in both, requires further explanation. The fact that these murmurs are heard as described is beyond question, but it seems much more reasonable to attribute the auricular (pulmonary?) murmur and the apex one to different and independent causes, as they are shown to be audible independently. I prefer to suppose that the so-called auricular murmur is in reality pulmonary, and that it is due to pressure on the pulmonary artery.

In further support of the opinion that these pulmonary systolic murmurs are produced by pressure on the pulmonary artery, I may refer to the widely-recognized fact that in certain instances of pleuritic effusion a basic systolic murmur is produced by the pressure of the fluid. Dr. Douglas Powell (*Diseases of the Lungs*, pp. 82, 83) thus describes it: "In cases in which the heart is greatly displaced by pleuritic effusion a systolic murmur is developed over its base, which disappears on removal of a portion of this fluid, and is presumably due to a straightening or slight tension of the great vessels from pressure of the fluid."

And as an example of the production of just such a murmur as we are considering, by direct pressure upon the pulmonary artery, I may refer to a case of "Aneurysm of the Aorta opening into the Pulmonary Artery" which I reported in the *Edinburgh Medical Journal*, February, 1888, which affords almost an experimental proof of it. In this case the pulmonary artery was pressed upon half an inch above the valves by a small aortic aneurism, which eventually communicated with it. Here there was marked pulsation in the third left intercostal space from the dilated conus arteriosus of the right ventricle and a loud systolic murmur. This was noticed before there was any ground for supposing that the opening of communication had formed, but while there was only pressure.

Before, however, this theory of the causation of the pulmonary systolic murmur by the pressure of an enlarged heart upon the pulmonary artery in the recumbent (supine) position can be accepted, it is necessary to show whether variations in bodily position influence the position and movements of the heart in the thorax. That is to say, whether the influence of gravity upon the heart does affect its pressure on neighboring organs. "In the healthy man, in the erect or sitting position, the heart, of course, lies on the diaphragm, suspended, however, a little by its base, which is attached by the great vessels and the structures of the spinal column. Owing to this arrangement of the parts it would seem, therefore, probable that variations in bodily position, or in the position of the diaphragm, might act through gravity upon the suspension of the heart, either lengthening it by allowing it to fall from its attachment, or shortening it by pushing it up and back, as it were, on its base." It is, of course, well known that the force and position of the apex-beat vary considerably in different positions of the body. But the influence of the position of the body on the position of the heart has been best shown by Mr. C. J. Bond, F.R.C.S., in some experiments both on the lower animals and on himself, a report of which was published in the *British Medical Journal*, December 12, 1885. By passing down the œsophagus an air tampon, connected by a tube with an air tambour and a recording apparatus, he was enabled to obtain tracings of the "base-beat" of the heart; and to show that it varied greatly in different positions of the body, and during inspiration and expiration. In describing a tracing taken while the tampon was in his own œsophagus, he says: "The smaller frequent rises (due to the cardiac beats) are more marked during expiration than inspiration, owing to the heart falling back toward the spine during expiration; further, these cardiac curves are very marked, especially on the summits of the expiratory rise at the left end of the tracing, while I was lying on my back with the head and shoulders depressed, and legs and pelvis raised, while they almost disappear in the sitting-up and prone positions, to reappear at the right-hand end of the tracing in the supine position."

In the next place, if the pressure theory has any foundation, why is not this pulmonary systolic murmur present in all cases of enlargement of the heart? In reply to this, I must say that a full explanation is wanting, but that it is present in some cases. I conceive a predominant dilatation of the right ventricle to be necessary, a condition not common in chronic heart disease. I have already mentioned that a systolic murmur with its point of maximum intensity in the second or third space, is not unfrequently audible in mitral stenosis—an affection which entails dilatation of the right ventricle, and not of the left.

I have also found it in the following case, which is typical of a class:

A man, aged forty-eight, has been under observation at frequent intervals during the past three years. He has chronic Bright's disease and a dilated (? fibroid) heart. When compensation fails, and he gets dropsy and cardiac symptoms with very rapid heart's action, a systolic murmur over the right and left ventricles is constant, and he has orthopnœa. When the heart is rehabilitated, and its speed reduced to seventy, the murmur disappears. Now he is just recovering from his fourth attack of failure of compensation, but the right side (and probably the left also) is considerably dilated. The cardiac dulness extends far to the right of the sternum, the jugulars are extremely turgid, pulsate with the heart-beats, and fill from below. The speed of the heart has been reduced to between eighty and ninety, but it is still irregular. In the erect position there is no trace of a murmur. In the recumbent position a systolic murmur is constantly audible, with its point of maximum intensity in the pulmonary area. This has been confirmed by several observers.

In cases of chronic valvular disease the changes are complex ; the left side is generally more enlarged than the right, and may prevent pressure on the pulmonary artery by transferring the weight of the heart to the aorta. Possibly, too, a certain degree of relaxation or flabbiness of the heart-muscle is required. When the left ventricle is considerably hypertrophied, and is firm and rigid, the much smaller right is more like an appendage to it, and cannot in the recumbent position fall back and press on the pulmonary artery.

As examples of the unfavorable influence of the dorsal recumbent position on the heart, where there is no suspicion of orthopnœa, I may quote the following, both of which have recently been under my observation:

A man, tall, thin, aged thirty-four, with a faint apex systolic murmur, and the heart's action regular and of normal speed, has some cardiac pain ; but is able to take active exercise and ride a bicycle. When he sleeps on his back he frequently wakes suddenly with urgent dyspnœa, and cardiac pain and distress.

A very stout, short man, aged fifty-two, with regular heart's action and no murmur, suffers from anginal attacks on walking up hill. If he lies on his back, cardiac pain always comes on within a few minutes, and continues until some time after he has turned on the right side, or has sat upright, or leaned forward, which latter position is the easiest.

Possibly, too, the distress and somewhat rapid fatal termination in sheep that are "cast," is due to the pressure on the heart from the dorsal position.

8. By this test of position the pulmonary murmur, when loudly audible in the other areas in the horizontal (supine) position, can be distinguished from true regurgitant murmurs due to relaxation of the heart-muscle, or dilatation of its openings, or valvulitis.

9. When in anœmia there are murmurs in all the areas, not due to permanent changes, they disappear during convalescence in a definite order, namely: first the tricuspid, next the mitral, then the aortic, and lastly the pulmonary.

With judicious treatment the murmurs, except the pulmonary, may cease in a fortnight to six weeks; but the pulmonary murmur, though less loud and distinct than heretofore, is generally audible in the horizontal, supine position for several weeks, and sometimes months, longer, notwithstanding an increase of weight on the part of the patient seven to fourteen pounds or more, and a corresponding improvement in color and general condition.

10. And lastly, it is probable that in some cases anœmic enlargement of the heart leads to permanent heart disease.

In order to learn whether anæmic enlargement of the heart ever leads to permanent heart disease, it is necessary to keep the cases under observation for some years, and that I have not been able to do. Of two cases that I have been able to examine at the end of two years or more, one had a permanent aortic systolic murmur, and the other a mitral systolic, but the pulmonary murmurs had gone. There was slight enlargement. It is not improbable that if more cases could be followed out permanent alterations would be found to be more common. Dr. Goodhart has suggested that there may be some connection between the great predominance of mitral stenosis in females, and the heart-changes of anæmia. He says: "Anæmia, by the dilatation it sets up, and the regurgitation through the mitral valve, may be the etiological factor in the unexplained considerable minority of cases of mitral stenosis in women which cannot be attributed to rheumatism."

11. Treatment.

If the importance and extent of the heart-changes are recognized the treatment is not difficult. It may be summed up in a few words: Rest, food, aperients, iron. Complete rest in bed for a week or ten days, with half the day in bed for another week or fortnight, is a therapeutic measure of great value. The dietary has generally been deficient in albuminoids. Saline aperients appear to answer best. I do not believe digitalis to be necessary except when the heart's action is too rapid even during rest, and in those cases which have to be treated without the possibility of rest. In the latter, better results may be obtained with digitalis and iron than by iron alone.